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THE EFFECTS OF MICROBIAL PESTICIDES ON NON-TARGET, BENEFICIAL ARTHROPODS

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ABSTRACT

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The formulation, testing protocol, lethal effects, sub-lethal effects, ecological relationships and selectivity of microbial pesticides on non-target, beneficial arthropod natural enemies of insect and mite pests are reviewed for viruses, bacteria, fungi and protozoa. In general, it was found that: (1) effects from indirect mortality of natural enemies are probably more significant across all microbial types than are direct mortalities from microbial pesticides; (2) significant levels of direct mortality of beneficial insects can be caused by bacterial and protozoan microbial pesticides; (3) no direct mortality from viruses of pests has been documented for arthropod natural enemies; (4) the direct effect of fungi on arthropod natural enemies has not been well studied and probably has been underestimated; (5) unique standard methods need to be developed for microbial assays and for expressing more exactly the dosage administered to the host or natural enemy.

INTRODUCTION

Use of microbial pesticides (MPs) for integrated pest management (IPM) has increased in recent years in part because of the inability to achieve selectivity to non-target species, such as biological control agents (i.e., arthropod predators and parasites), with conventional organosynthetic pesticides (Mullin and Croft, 1985). Pathogenic viruses, bacteria, fungi and protozoa are currently being tested to determine their selectivity, safety and economic viability for IPM. As genetic engineering of insect pathogens nears the forefront of biotechnological research, an array of "engineered pathogens" showing new spectra of activity and selectivity should be forthcoming for experimental testing and evaluation (Kirschbaum, 1985).

To date, relatively few conventional or naturally occurring MPs have been commercially developed, registered and marketed successfully. As most MPs have characteristics readily amenable to the production and marketing technologies of conventional pesticides, there has been a tendency to view

assessment of MP impact on non-target species and protocols for registration in ways similar to those adopted for conventional chemical pesticides. Some scientists contend that since many microbials occur in nature without harmful effects to non-target species of concern to man, they should not require such extensive evaluation or registration requirements before use is permitted. Other professionals note that as some of these microorganisms are able to persist and replicate in nature, they should require stricter evaluations, especially where feedback effects on host species populations operating in the environment may continue for many generations.

These extremes of perspective and the questions they raise regarding impact on non-targets are not simple to answer. It is likely that both philosophies contain elements of truth, i.e. there are certain microbials that are safer and have fewer side effects to non-target species than conventional pesticides, but there may be others with more complex, long-term effects on non-target species that cannot be assessed by conventional impact assessment techniques. The critical question is which type are we dealing with in a specific case, and what additional assessment should be developed to evaluate these agents, especially in light of new types of microbial agents likely to be "genetically engineered" in the future?

In this review, we summarize the status of testing methods and impact studies of microbial pesticides on non-target beneficial arthropods, focusing primarily on predator and parasite natural enemies of agricultural pests. These arthropods are important components of pest management as biological control agents on major crops grown worldwide (Hoy and Herzog, 1985). Although considerable information on other non-entomophagous beneficial insects such as the honey bee (*Apis mellifera*) and silk worm moth (*Bombyx mori*) are available, these are not treated extensively here (see reviews for these species in Cantwell et al. (1966), Cantwell and Lehnert (1979) and Forsberg et al. (1976). Individual sections on viruses, bacteria, fungi and protozoa are presented; within each of these sections, the available literature on formulations used, methods of assessment and lethal and sub-lethal impacts are summarized. In a final section, current testing procedures for measuring the impact of microbials on non-target species are critically reviewed. We speculate on the expanded needs of assessment methods for new biotechnologically designed microbials on non-target beneficials and point out the useful role that these arthropod models may play in evaluating the impact of genetically engineered agents, in general (including those other than microbial pesticides).

VIRUSES

Viruses are sub-microscopic particles with double or single strands of RNA or DNA that persist and replicate as obligate intracellular parasites. Their physiological "machinery" operates only upon host content, i.e.

infection. In insects, virus infection is via ingestion, integumental wounds, transovarial or transovum transmission. The virus then attaches to specific cell membrane sites in the host, where penetration of the cell membrane is initiated. After penetration, the virus incorporates its genetic material with that of its host to replicate and ultimately release new viral propagules. Release may be intercellular, spreading the infection within the host, or infection may be spread between hosts by the release of free particles released in fecal droppings, or the disintegrating carcasses of the host. The duration from infection to death in insects is dependent upon the size of the inoculum and virulence of the virus, but usually ranges from 5 to 20 days or longer (Cherwonogrodzky, 1980). Reviews of general aspects of virology may be found in Fraenkel-Conrat and Kimball (1982), or aspects of insect virology in Burges and Hussey (1971), Burges (1981), Cherwonogrodzky (1980), or Kurstak (1982).

The majority of the economically important entomopathogenic viruses are DNA viruses found in the family Baculoviridae genus *Baculovirus*. These include the nuclear polyhedrosis viruses (NPV) in Subgroup A that contain multiple virions embedded in a proteinaceous occlusion body, the granulosis viruses (GV) in Subgroup B that contain a single virion in an occlusion body and the non-occluded viruses in Subgroups C and D. These viruses mainly infect mid-gut, trachea, fat and epidermal cells. The cytoplasmic polyhedrosis viruses (CPV) in the family Reoviridae and entomopoxviruses in the family Poxviridae may also have potential in microbial control of insect pests. Viruses in other families, such as the Nodaviridae, are not being commercially considered because they are closely related to viruses that infect vertebrates.

The majority of lethal response tests of entomopathogenic viruses on non-target beneficial arthropods have been conducted on the predators and parasites of lepidopterous pests. Table I lists, by family, many of the beneficial insects that have been tested with entomopathogenic viruses for lethal or sub-lethal effects. All tests listed are with different strains of NPV or GV within the genus *Baculovirus*.

Formulation

Production and registration of commercial entomopathogenic viruses has been slow, primarily because the high degree of host specificity of these organisms make them relatively unattractive economically to manufacture. However, experimental spray programs have been conducted in both the United States and Canada for several important forest insect pests (Kingsbury et al., 1977; Brooks et al., 1978; Doane and McManus, 1981). One of the few commercial formulations, Viron/H[®], has been economically successful partially because it has a relatively broad host range (i.e. it infects six species

TABLE I

Effects of viral¹ pathogens on non-target insects

Non-target insect		Virus pathogen	Virus dosage	Method of exposure
Family	Species			
Apidae	<i>Apis mellifera</i>	NPV	2.6 and 6.3 $\times 10^6$ pib ml ⁻¹	Ingested by adults
		GV	1.5×10^6 granula ml ⁻¹	Ingested by adults
		NPV	5×10^7 pib ml ⁻¹ 1.7×10^8 pib g ⁻¹ (wp)	Contact and ingested by adults
		GV	1×10^{10} granula ml ⁻¹ 1×10^{11} granula g ⁻¹ (wp)	Contact and ingested by adults
		NPV	5.5 billion pib ha ⁻¹	Aerial application
		NPV	7.5×10^4 and 7.5×10^5 pib ml ⁻¹	Ingested by adults
Braconidae	<i>Chelonus blackburni</i>	NPV (Viron/H®)	Minimum recommended field dose	Contact by adults
	<i>Chelonus insularis</i>	HGV (Hawaiian)	Varied to cause frank infection of the host before emergence	Ingested by larvae within host
	<i>Cotesia glomeratus</i>	HNPV (hypertrophy)	1.0 $\times 10^5$ granula ml ⁻¹	Ingested by host larvae after being parasitized
		GV		
	<i>Cotesia marginiventris</i>	HGV (Hawaiian)	Varied	Ingested by larvae in host
	<i>Glyptapanteles militaris</i>	NPV (typical)	6.4×10^{10} pib ml ⁻¹	Ingested by host larvae 3-8 days after being parasitized
		HNPV (hypertrophy)	1.4×10^9 pib ml ⁻¹	
		HGV (Hawaiian)	1.8×10^{10} gram ml ⁻¹	

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
<i>Autographa californica</i>	*	Lab.	Cantwell and Lehnert, 1979	—	—
<i>Heliothis virescens</i>	*	Lab.		—	
<i>Mamestra brassicae</i>	*	Lab.	Groner et al., 1978	—	24–240 h
<i>Cydia pomonella</i>	*	Lab.		—	24–240 h
<i>Neodiprion lecontei</i>	*	Fld.	Kingsbury et al., 1977	—	1440 h
<i>A. californica</i>	*	Lab.	Morton et al., 1975	—	1–120 h
<i>Heliothis</i> sp.	*	Lab.	Wilkinson et al., 1975	—	—
<i>Spodoptera exigua</i>	*	Lab.	Hotchkin and Kaya, 1983a	—	—
<i>Pseudaletia unipuncta</i>	****1			IDT	—
<i>Pieris rapae</i>	**** (< 5 days)			—	—
	* (> 8 days)	Lab.	Levin et al., 1981		
<i>S. exigua</i>	**	Lab.	Hotchkin and Kaya, 1983a	—	—
<i>P. unipuncta</i>	*				
<i>P. unipuncta</i>	*	Lab.	Hotchkin and Kaya, 1983b	—	—
	****			—	—
	****		Kaya and Tanada, 1972a		

TABLE I (continued)

Non-target insect		Virus pathogen	Virus dosage	Method of exposure
Family	Species			
	<i>Meteorus leviventris</i>	NPV Viron/H®	Minimum recommended field dose	Contact by adults
	<i>Phanerotoma flavitestacea</i>	HGV	1 µl of infected plasma	Injected into parasitized host larvae
Chalcidae	<i>Brachymeria intermedia</i>	NPV (Viron/H®)	Minimum recommended field dose	Contact by adults
Ichneumonidae	<i>Campoletis sonorensis</i>	HGV (Hawaiian)	Varied for frank infection	Ingested by larvae in host
		HNPV (hypertrophy) NPV	5 × 10 ⁶ pib ml ⁻¹	Ingested by larvae in host
				<i>Heliothis</i> exposed to virus 24, 36, 48, 60 and 72 h after parasitization
				Contact by adults
	<i>Devorgilla canescens</i>	NPV (Viron/H®)	Minimum recommended field rate	Contact by adults
		HGV GV	1 µl of plasma	Ingested by larvae in host
Ichneumonidae	<i>Hyposoter exiguae</i>	NPV	1 × 10 ⁵ pib ml ⁻¹	Ingested by larvae in host. <i>T. ni</i> exposed ≤ 3 days before and 6 after parasitization
		HNPV HGV (Hawaiian)	Varied	Ingested by larvae within host
Trichogrammatidae	<i>Trichogramma cacoeciae</i>	NPV	1, 5 and 10 XLC ¹⁰⁰ for <i>M. brassicae</i>	Contact and ingested by adults and sprayed on eggs

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
<i>Heliothis</i> sp.	*	Lab.	Wilkinson et al., 1975	—	1–120 h
<i>Anagasta kuehniella</i>	***	Lab.	Kaya and Tanada, 1972a	—	129–216 h
<i>Heliothis</i> sp.	*	Lab.	Wilkinson et al., 1975	—	1–120 h
<i>S. exigua</i> <i>P. unipuncta</i>	*	Lab.	Hotchkin and Kaya, 1983a	IDT	—
				—	—
<i>H. virescens</i>	**** (< 36 h) *** (48 h) ** (60 h) * (72 h)	Lab.	Irabagon and Brooks, 1974	—	—
<i>Heliothis</i> sp.	*	Lab.	Wilkinson et al., 1975	—	1–120 h
<i>A. kuehniella</i>	*	Lab.	Kaya and Tanada, 1972a	—	—
<i>Trichoplusia ni</i>	**** (< 48 h) *** (72–96 h) ** (120–148 h)	Lab.	Beegle and Oatman, 1975	—	192–216 h
<i>S. exigua</i> <i>P. unipuncta</i>	*	Lab.	Hotchkin and Kaya, 1983a	—	—
<i>Mamestra brassicae</i>	*	Lab.	Hassan and Groner, 1977	IDT	—

TABLE I (continued)

Non-target insect		Virus pathogen	Virus dosage	Method of exposure
Family	Species			
	<i>Trichogramma euproctidis</i> and <i>Trichogramma evanescens</i>	GV NPV	— —	Sprayed on <i>Sitotroga cerealella</i> eggs before parasitization
Coccinellidae	<i>Hippodamia convergens</i>	NPV (Viron/H®)	Minimum field rate	Contact by adults
Tachinidae	<i>Comptosia concinnata</i>	HGV (Hawaiian) HPNV (hyper- troph)	Varied	Ingested by larvae in host
	<i>Para-sarcophaga misera</i>	NPV	—	Ingested by larvae in host
	<i>Voria ruralis</i>	NPV (Viron/H®)	Field rate	Contact by adults
		NPV	—	Ingested by larvae in host
Chrysopidae	<i>Chrysopa carnea</i>	NPV NPV (Viron/H®)	1, 5 and 10 x LC ¹⁰⁰ for <i>M.</i> <i>brassicae</i> Field rate	Ingested and contacted by larvae Contact by adults

* = (0-10% mortality) no or slight toxicity.

** = (10-40% mortality) low toxicity.

*** = (40-80% mortality) moderate toxicity.

**** = (80-100% mortality) high toxicity.

¹ *Baculovirus* subgroup A — NPV; *Baculovirus* subgroup B₁ — GV.

² Host died before larvae completed development.

‡ IDT = increased developmental time.

of *Heliothis* and *Helicoverpa*) (Burgess and Hussey, 1971). Currently, entomopathogenic viruses must be grown on living hosts which is a labor intensive, costly operation. However, as advances in tissue culture production and genetic engineering are made in this area and if public demand increases for microbial pesticide production, then the economic feasibility of manufacturing commercially available entomopathogenic viruses should increase.

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
	*		Vybornov et al., 1982	—	—
<i>Xestia c-nigrum</i>	*	Lab.		—	—
<i>Heliothis</i> sp.	*	Lab.	Wilkinson et al., 1975	—	1-120 h
<i>P. unipuncta</i>	*	Lab.	Hotchkiss and Kaya, 1983a	—	—
<i>Spodoptera litura</i>	*	Lab.	Battu, 1977 Battu and Dilawari, 1978	—	—
<i>Heliothis</i> sp.	*	Lab.	Wilkinson et al., 1975	—	1-120 h
<i>T. ni</i>	*	Lab. Fld.	Vall, 1981	—	—
<i>M. brassicae</i>	*	Lab.	Hassan and Groner, 1977	—	—
<i>Heliothis</i> sp.	*	Lab.	Wilkinson et al., 1975	—	1-120 h

Testing protocol

In judging the appropriateness and applicability of side effects information available for a particular predator or parasitoid and microbial agent, it is important to examine how the test organism was exposed and related features of the methodology. In 35% of the tests (Table I), direct contact toxicity to the virus was assessed. Mortality was very low (never exceeding 4%) when five parasitoids and two predators of *Heliothis* were tested for

contact toxicity of Viron/H® (Wilkinson et al., 1975). Low contact toxicity would be expected since viruses only alter tissues found in the mid-gut epidermis or fat body. They must be ingested or penetrate into the hemocoel to have an effect on their host.

The majority of the beneficial insects assayed with entomopathogenic viruses were tested for acute effects after they had ingested the virus (Table I). Adults were fed known concentrations of polyhedral inclusion bodies (pib) or granules per milliliter in a contaminated food source where uptake could be monitored and quantified (Morton et al., 1975; Hassan and Groner, 1977; Groner et al., 1978; Cantwell and Lehnert, 1979). Direct toxicity in these bioassays was all very low, suggesting that there are no acute effects to adults. Other feeding tests were conducted on larval endoparasitoids, where actual amounts of the virus consumed while feeding on the host could not be easily quantified (Table I). Tests were run either by infecting a pre-parasitized host larva with a virus or by allowing a female parasitoid to oviposit in a pre-infected host. Quantifying the effect of the viruses on the parasitoid larvae was difficult, because all deleterious effects to the parasitoid larvae were indirect, caused by either killing or physiologically altering the host (Hotchkiss and Kaya, 1983a).

Duration of tests with viruses varied. Many of the assays listed in Table I monitored the infected host until it died or the parasitoid emerged. Other tests were run with chronological endpoints ranging from 3 to 60 days. Some articles gave no information on test duration.

Lethal effects

No documentation of direct mortality, of any arthropod parasitoid or predator, induced by infection from a host baculovirus has been cited in existing literature (Hotchkiss and Kaya, 1983a). However, indirect mortality of parasitoid larvae can occur (Table I). Immature parasitoids can die if a virus renders their host unsuitable before the larvae can mature. This usually occurs if the host dies before the parasitoid completes its development (Irabagon and Brooks, 1974; Beegle and Oatman, 1975; Levin et al., 1981). Indirect mortality may also be due to physiological alterations of the host which can produce toxic byproducts (Kaya, 1970; Kaya and Tanada, 1971a,b, 1972a,b, 1973; Hotchkiss and Kaya, 1983a,b).

Mortality of the ichneumonid *Campoletis sonorensis* was 96% when its host, *Heliothis virescens*, was infected, with 4×10^6 polyhedral inclusion bodies (pib) ml^{-1} of NPV, 24 h after being parasitized. Mortality decreased to 49% when *H. virescens* was infected 48 h after being parasitized and 6% when infected 72 h after oviposition (Irabagon and Brooks, 1974). Similarly, Beegle and Oatman (1975) found that all *Hyposoter exiguae* that parasitized *Trichoplusia ni* which had been infected 1–3 days earlier with 1×10^5 pib ml^{-1} of NPV, died when *t. ni* died. Ninety-two percent of the ichneumonids died if parasitized *T. ni* larvae were infected with the virus 48 h

after oviposition, 56% died if infected after 96 h and 20% when infected 144 h after oviposition. Similar results are reported for the braconid, *Cotesia glomeratus*, which suffered 100% mortality if its host, *Pieris rapae*, was infected with 10^5 GV granules⁻¹ ml⁻¹ 96 h after parasitization. Mortality decreased linearly and was approximately 10% when the host was infected 216 h after oviposition (Levin et al., 1981).

To summarize, parasitoid survival and emergence in a virus-infected host can be significantly affected by the timing of exposure of the host to the virus. Parasitoid survival increased as the period between oviposition and virus infection increased (Levin et al., 1981). Thus, percent survival of parasitoids emerging from virus infected hosts is dependent on the developmental rate of the virus, as well as the developmental rate of the larvae.

Another cause of indirect mortality of parasitoid larvae is physiological alteration of the host. This has been shown to occur during replication of the Hawaiian strain of granulosis virus (HGV) or the hypertrophy strain of nuclear polyhedrosis virus (HNPV) which produce toxic factors that adversely affect the development of *Glyptapanteles militaris* (Kaya, 1970; Kaya and Tanada, 1972a,b; Hotchkiss and Kaya, 1983a,b, 1985). When *Pseudaletia unipuncta* were parasitized by the braconid *G. militaris* and fed the HGV within 24 h, 91% of the parasitoids died in the first instar. Conversely, only 6% of the *G. militaris* died when *P. unipuncta* was fed a different strain of GV (Kaya and Tanada, 1972b). Kaya and Tanada (1972a) report a similar effect on *Phanerotoma flavitestacea* when its host, *Anagasta kueiella*, was infected with the HGV. A hypertrophy strain of NPV (HNPV) also killed 100% of the *G. militaris* in the first or second instar when fed to its host, *P. unipuncta*; yet parasitoids within *P. unipuncta* infected with a typical strain of NPV were in the second instar and appeared normal (Kaya and Tanada, 1973). When viron-free plasma taken from armyworms infected with the hypertrophy strain was injected into uninfected, parasitized armyworms, the development of the parasitoid was adversely affected, suggesting that the viron of the hypertrophy strain is not the toxic factor (Kaya and Tanada, 1973). Hotchkiss and Kaya (1983b) tested the toxic effect of the HGV and HNPV on one tachinid, two ichneumonids and three braconids and found that only *G. militaris* died in HNPV-infected *P. unipuncta* before the host died. The braconid *Chelonus insularis* died after its HGV- or HNPV-infected host died, but this high mortality probably occurred because *P. unipuncta* died before *Chelonus* completed its development within this factitious host (Hotchkiss and Kaya, 1983b).

Sub-lethal effects

The majority of tests measuring sub-lethal effects (Table I) on adult and larval parasitoids and predators have given results not significantly different from the controls (Irabagon and Brooks, 1974; Hassan and Groner, 1977; Levin et al., 1981; Hotchkiss and Kaya, 1983a). However, significant effects

on developmental time of parasitoid larvae within virus-infected hosts have been reported (Beegle and Oatman, 1975; Kaya and Hotchkin, 1983a).

There were no adverse effects on the larval feeding capacity, adult fecundity or egg viability of *Chrysopa carnea* when adults were sprayed directly, or larvae were fed *Mamestra brassicae* infected with NPV (Hassan and Groner, 1977). Hassan and Groner (1977) also tested *Trichogramma cacoeciae* and found no reduction in their parasitism rate or development after the parasitoid had been exposed to or ingested the NPV of *M. brassicae*. Beegle and Oatman (1975) found no significant difference in pupal developmental time, adult emergence, adult longevity or percent parasitism between *H. exiguae* that emerged from infected and noninfected *T. ni*. However, developmental time of those *Hyposoter* that successfully emerged from parasitized *T. ni* was significantly shorter than those emerging from uninfected hosts. This difference may have resulted from an actual decrease in the developmental time of the parasitoids within infected hosts, or the mean developmental time was reduced because slower developing parasitoid larvae died when their host died from viral infection (Beegle and Oatman, 1975).

Hotchkin and Kaya (1983a) also found that *H. exiguae* had a shorter, but not significant, developmental time within HNPV-infected *P. unipuncta*. In general, although, their study showed that hymenopterous parasitoids from infected hosts took a longer, not shorter, time to develop and emerge than parasitoids developing in uninfected hosts. They suggest that this longer developmental time is related to the nutritional quality of the host because viral infection reduced host size which provides insufficient nutrition for the developing parasitoid larvae (Hotchkin and Kaya, 1983a).

Ecological relationships and selectivity

In general, baculoviruses have a narrow host range and no evidence of direct toxicity to natural enemies has been documented (Table I). All lethal and sub-lethal effects are indirect with the majority associated with host unsuitability due to viral infection. All lethal effects reported are confined to immature parasitoids developing within virus-infected hosts. Mortality was greatest when parasitoids oviposited in virus-infected hosts and decreased as the period between oviposition and host-virus infection increased (Levin et al., 1981). Thus, the ability to discriminate virus-infected hosts from healthy hosts should provide a strong selective advantage for parasitoids of species that exhibit periodic viral epizootics.

Host discrimination due to viral infection has been documented for a parasitoid of the gypsy moth, *Lymantria dispar* (Versoi and Yendol, 1982). *Cotesia melanoscelus* females can discriminate between non-infected and NPV-infected gypsy moth larvae. The percent of ovipositional attempts in the non-infected larvae was 68.7%, which was significantly greater than the virus-infected larvae ($P < 0.01$) which was only 32.1% (Versoi and Yendol, 1982). This implies that the parasite does not "waste" eggs on a host which

is soon to die (Hotchkiss and Kaya, 1983a). Thus, if the parasitoids can discriminate between healthy and infected hosts in the field, the actual impact on beneficial non-targets may be less than predicted from laboratory experiments, where parasites oviposit in viral-infected host only because they are "host starved" and have no option to lay their eggs anywhere else.

BACTERIA

The most widely used and commercially available microbial pesticides are bacteria. Commercial production, however, has been restricted primarily to one genus of bacteria, *Bacillus*, and to strains and varieties of only two species: *Bacillus thuringiensis* (*B. t.*) and *B. popilliae* (*B. p.*). Production of *B. t.* began in the U.S.A. after the second World War and by the mid 1960's it was being used to protect 50% of the cole-crops (brassicas) in Southern California from *Trichoplusia ni* (Burgess and Hussey, 1971). Today it has been used to control over 40 species of lepidopterous pests, mosquitoes and black flies with hundreds of tons of spore-products being manufactured each year in at least five different countries (Cherwonogrodzky, 1980).

Bacillus thuringiensis is a gram-positive, aerobic, spore forming bacterium whose vegetative cell becomes a sporangium producing a spore and parasporal protein crystal (Burgess and Hussey, 1971; Cherwonogrodzky, 1980). Death of susceptible insects can occur from ingestion of the spores, crystals or exotoxins. An insect must have a strongly alkaline mid-gut (pH of 9.0–10.5), specific proteolytic enzymes and suitable tissue receptor-sites to be susceptible (Cherwonogrodzky, 1980). Thus, many non-target insects are not affected by this bacterium. However, some evidence suggests that *B. thuringiensis* may induce mortality in beneficial non-target arthropods (Hamed, 1979; Muck et al., 1981; Tanada, 1984).

With the exception of the tests done on the honeybee, *Apis mellifera* and the tests conducted with *Bacillus thuringiensis* var. *israelensis* (*B. t. i.*) on non-target aquatic insects, the majority of lethal response tests of bacterial pathogens on non-target arthropods have been conducted on predators and parasites of lepidopteran pests. Table II lists, by family, many of the beneficial insects that have been tested with bacterial pathogens for lethal effects. This table does not include any of the lethal concentration tests conducted on non-target aquatic organisms with *B. t. i.* Most of these tests have shown there are no measurable direct effects of *B. t. i.* on any of the numerous species of aquatic insects tested, with the exception of the dipterous families Chironomidae, Dixidae and Ceratopogonidae which are moderately to severely affected (Garcia et al., 1980; Sinegre et al., 1980; Ali, 1981; Mulla, 1982; Mulligan and Schaefer, 1982; Seckinger, 1982). Table II, also, does not include the majority of the work done on honey bees or a list of beneficial lepidopteran affected by *B. t.* For a review of the earlier tests conducted on *Apis mellifera* see Cantwell et al. (1966) and Cantwell et al. (1972). A partial list of beneficial lepidoptera affected by *B. t.* may be found in Forsberg et al. (1976).

TABLE II

Effects of bacterial pathogens on non-target arthropods

Non-target insect		Bacteria pathogen	Bacteria dosage	Method of exposure
Family	Species			
Aphelinidae	<i>Encarsia formosa</i>	Entobakterin (B. t. var. <i>galleriae</i>)	—	—
	<i>Aphytis melinus</i>	B. t. + surfactant	3760 ppm	Residue (on scale cover)
Apidae	<i>Apis mellifera</i>	Thuricide-HP (B. t. var. <i>kurstaki</i>)	9×10^8 spores g^{-1}	Ingested by small larvae
			0.2×10^8 spores g^{-1}	
			30×10^8 spores g^{-1}	
		<i>Bacillus sphaericus</i>	5.4×10^4 spores g^{-1}	Ingested by adults
			5.4×10^6 spores g^{-1}	
			8.7×10^7 spores g^{-1}	
		<i>Bacillus moritai</i>	4×10^8 spores ml^{-1}	Ingested by adults
Braconidae	<i>Agathis rufipes</i> <i>Apanteles fumiferanae</i>	Entobakterin	0.5% a.i.	Ingested by adults
		Dipel WP®	453 g/7.61/0.4/ha	Ingested by larvae in host
		Thuricide®	7.6 BIU $acre^{-1}$ 8 BIU $acre^{-1}$	
	<i>Ascogaster quadridentata</i>	Entobakterin®	0.5% a.i.	Ingested by adults
	<i>Bracon brevicornis</i>	Bakthane® B. t. var. <i>thuringiensis</i>	3×10^{10} spores ml^{-1}	Ingested by larvae in host
	<i>Cardiochiles nigriceps</i>	Dipel WP® Biotrol XK®	16,000 IU mg^{-1} 2 $mg\ ml^{-1}$	Ingested by adults contact by adults
	<i>Cotesia melanoscelus</i>	Dipel HG® (B. t. var. <i>kurstaki</i>)	9.3–75.0 IU ml^{-1}	Ingested by larvae in host
		Dipel 4H®	19.8 BIU ha^{-1}	
		Dipel®	2.0×10^{10} IU ha^{-1}	

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
<i>Trialeurodes vaporariorum</i>	*	Lab.	Beglyarov and Maslienko, 1978	—	—
<i>Aonidiella aurantii</i>	*	Lab.	Davies and McLaren, 1977	—	96-960 h
—	*** *** ****	Lab.	Ali et al., 1973	—	—
—	* * *	Lab.	Cantwell and Lehnert, 1979; Davidson et al., 1977	— — —	— — —
—	*	Lab.	Ozino et al., 1972	—	48 h
—	*	Lab.	Krieg et al., 1980	—	—
<i>Cydia pomonella</i>	*	Lab.	Mosievskaya and Makarov, 1974	—	—
<i>Choristoneura occidentalis</i>	*	Fld.	Hamel, 1977	—	—
<i>C. occidentalis</i>	*	Fld.	Thompson et al., 1977	—	—
<i>C. pomonella</i>	*	Lab.	Mosievskaya and Makarov, 1974	—	—
<i>Sesamia cretica</i>	**	Lab.	Temerak, 1980 and Temerak, 1982a, b	REP, RAL IDT	—
<i>Heliothis virescens</i>	***	Lab.	Dunbar and Johnson, 1975;	RAL	48- 192 h
	*	Lab.	Wilkinson et al., 1975	—	1-120 h
<i>Lymantria dispar</i>	*	Lab.	Ahmad et al., 1978	—	186- 336 h
	*			IPP	—
	*	Fld.	Ticehurst et al., 1982;	IPP	—
	*	Fld.	Weseloh et al., 1983	IPP	—

TABLE II (continued)

Non-target insect		Bacteria pathogen	Bacteria dosage	Method of exposure
Family	Species			
	<i>Cotesia glomeratus</i>	Bactospeine® (<i>B. t.</i> var?) Dipel®	0.05% a.i. 1×10^8 spores ml^{-1} 1×10^9 spores ml^{-1}	Ingested by adult Ingested by adult
	<i>Meteorus leviventris</i>	Thuricide® HPC	Field rate	Contact by adults
	<i>Microgaster rufipes</i>	Bitoxibacillin 202®	70 milliard g^{-1}	—
	<i>Microplitis demolitor</i>	<i>B. t.</i> var <i>entomocidus</i>	500 $\mu\text{g ml}^{-1}$	Ingested by larvae in host
	<i>Phanerotoma flavitesticata</i>	Bactospeine®	700 iu mg^{-1}	Contact of adult and ingested by larvae in host
	<i>Rogas tymantriae</i>	<i>B. t.</i> strain HD-1S-1980	24 IU ml^{-1}	Ingested by larvae in host
Chalcididae	<i>Brachymeria intermedia</i>	Thuricide HPC®	Recommended field rate	Contact of adult
Encyrtidae	<i>Ageniaspis fuscicollis</i>	b-exotoxin of <i>B. t.</i> var <i>thuringiensis</i> Dipel®	— 1×10^8 spores ml^{-1}	Contact by adult Ingested by adult
	<i>Leptomastix dactylopii</i>	Dipel®	0.10% a.i.	Ingested by adults Contact of adult
Eulophidae	<i>Tetrastichus evonymellae</i>	<i>B. t.</i> var <i>kurstaki</i>	1×10^8 spores ml^{-1} 5×10^8 spores ml^{-1}	Ingested by adult
Formicidae	<i>Formica pratensis</i>	BTB-202®	0.5% a.i. 1.0% a.i. 1.5% a.i.	Ingested by adults
Ichneumonidae	<i>Agrypon</i> sp.	<i>B. t.</i> var <i>kurstaki</i>	1×10^8 spores ml^{-1} 5×10^8 spores ml^{-1}	Ingested by adults
	<i>Camponotus sonorensis</i>	Thuricide HPC®	Recommended field rates	Contact by adults
	<i>Diadegma armillata</i>	<i>B. t.</i> var <i>kurstaki</i>	1×10^8 spores ml^{-1}	Ingested by adults
	<i>Exeristes comstockii</i>	<i>Serratia marcescens</i>	2–20% infection of host	Ingested by larvae in host

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
<i>Pieris brassicae</i>	**	Lab.	Marchal-Segault, 1975a	RHC, IDT, —	
	**	Lab.	Muck et al., 1981	RAL —	336 h

—	*	Lab.	Wilkinson et al., 1975	—	1-120 h
<i>C. pomonella</i>	*	Fld.	Kazakova and Dzhunusov, 1977	—	—
<i>Spodoptera littoralis</i>	****	Lab.	Salama et al., 1982	REP	—
<i>Ephestia kuehniella</i>	**	Lab.	Marchal-Segault, 1975a, b, c	RHC, IDT, — RAL	
<i>L. dispar</i>	*	Fld.	Wallner et al., 1983	IPP, SSR	—
—	*	Lab.	Wilkinson et al., 1975	—	—
—	*	Lab.	Korostel and Kapustina, 1975	—	—
<i>Yponomeuta evonymellus</i>	**	Lab.	Hamed, 1979	—	—
—	**	Lab.	Franz et al., 1980	—	—
	*	Lab.		—	—
<i>Y. evonymellus</i>	**	Lab.	Hamed, 1979	—	—
<i>P. brassicae</i>	*	Lab.	Legotai, 1980	—	—
<i>Y. evonymellus</i>	*	Lab.	Hamed, 1979	—	1-120 h
—	*	Lab.	Wilkinson et al., 1975	—	—
<i>Y. evonymellus</i>	**	Lab.	Hamed, 1979	—	—
<i>Galleria mellonella</i>	****	Lab.	Bracken and Bucher, 1967	—	—

TABLE II (continued)

Non-target insect		Bacteria pathogen	Bacteria dosage	Method of exposure
Family	Species			
	<i>Glypta fumiferanae</i>	Dipel®	453 g/7.61/0.4 ha	Contact by adults
		Dipel®	7.6 BIU acre ⁻¹	Ingested by larvae in host
		Thuricide®	8 BIU acre ⁻¹	
	<i>Phobocampe uncinata</i>	Dipel 4H®	19.8 BIU ha ⁻¹	Ingested by larvae?
	<i>Phygadeuon trichops</i>	Dipel®	0.10% a.i.	Contact by adults
		Dipel®	—	Contact by adults
	<i>Pimpla instigator</i>	<i>B. t. var thuringiensis</i>	—	Ingested by adults and larvae in host
	<i>Pimpla turionellae</i>	Dipel®	1 × 10 ⁷ 1 × 10 ⁹	Ingested by adults
	<i>Zele chlorophthalma</i>	<i>B. t. var entomocidus</i>	500 µg ml ⁻¹	Ingested by larvae in host
Scelionidae	<i>Telenomus alsophilae</i>	Biotrol XKW® Dipel WP® Thuricide HPC®	0.32, 0.64 lb ga ⁻¹ 0.10, 0.20 lb ga ⁻¹ 0.40, 0.80 lb ga ⁻¹	Contact from exposed egg masses
Trichogrammatidae	<i>Trichogramma cacoeciae</i>	Dipel®	0.10% a.i.	Contact by adult
		Dipel®	54.7 × 10 ⁹ spores g ⁻¹	
		Thuricide®	50.7 × 10 ⁹ spores g ⁻¹	
		Bactospeine®	1.0 × 10 ⁹ spores g ⁻¹	Contact and ingested by adults and larvae
		Entobakterin®	1.0% a.i.	Contact by adults
	<i>T. embryophagum</i>	Entobakterin®	1.0% a.i.	Contact by adults
		Entobakterin®	0.6% a.i.	Contact by adults
	<i>T. evanescens</i>	Entobakterin®	1.0% a.i.	Contact by adults
		BTB 202®	0.5, 1.0, 1.5% a.i.	Ingested by adults

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
<i>C. occidentalis</i>	*	Fld.	Hamel, 1977	—	—
<i>C. occidentalis</i>	*	Fld.	Thompson et al., 1977	—	—
<i>L. dispar</i>	*		Ticehurst et al., 1982	—	—
—	*	Lab.	Franz et al., 1980	—	—
—	*	Lab./ Fld.	Naton, 1978	—	—
<i>P. brassicae</i>	*	Lab.	Biache, 1975	—	432 h
—	* **	Lab.	Muck et al., 1981; Hamed, 1979	—	—
<i>S. littoralis</i>	***	Lab.	Salama and Zaki, 1983	RPE, RPP, IDT	—
<i>Ennomos subsignarius</i>	*	Lab./ Fld.	Kaya and Dunbar, 1972	—	240 h
—	*	Lab.	Franz et al., 1980	—	—
<i>Sitotroga cerealella</i>	*			REP	—
	*	Lab.	Hassan and Krieg, 1975		
—	*	Fld.	Kapustina, 1977	—	—
—	*	Fld.	Kapustina, 1977	—	—
<i>C. pomonella</i>	*	Lab.	Kostadinov, 1979	—	24 h
—	*	Fld.	Kapustina, 1977	—	—
<i>P. brassicae</i>	*	Lab.	Legotai, 1980	—	—

TABLE II (continued)

Non-target insect		Bacteria pathogen	Bacteria dosage	Method of exposure
Family	Species			
	<i>T. pallidum</i>	Dipel®	1.0% a.i.	Contact of larvae and ingested by larvae
		Entobakterin®	1.0% a.i.	
		Toxobakterin®	1.0% a.i.	
		Exobakterin®	1.0% a.i.	
	<i>Trichogramma</i> sp.	Bitoxibacillin 202®	70 milliard g ⁻¹	—
	<i>Trichogramma</i> sp.	<i>B. t. var thuringiensis</i>	—	Contact by adults ingested by adults
Carabidae	<i>Trichogramma</i> sp.	Dendrobacillin®	—	Adults exposed to residue
	<i>Bembidion lampros</i>	Dipel®	1.12 kg ha ⁻² five times during season	Contact by adults
			0.1% a.i.	Contact by adults
			0.5% a.i.	Contact by adults
Coccinellidae	<i>Adonia variegata</i>	Dendrobacillin®	2.0% a.i.	Contact by adults
		Entobakterin®	2.0% a.i.	Ingested by adults
	<i>Coccinella septempunctata</i>	Bitoxibacillin 202®	70 milliard g ⁻¹	—
		Dipel®	1.0% a.i.	Ingested by larvae
		Entobakterin®	1.0% a.i.	
		Ecotoxin®	1.0% a.i.	
		Toxobakterin®	1.0% a.i.	
		BTB-202®	0.5, 1.0, 1.5% a.i.	Ingested by adults
		Dendrobacillin®	2.0% a.i.	
		Entobakterin®	2.0% a.i.	
	<i>Coccinella undecimpunctata</i>	<i>B. t. var entomocidus</i>	500 µg ml ⁻¹	
		Dendrobacillin®	2.0% a.i.	Contact by adults
		Entobakterin®	2.0% a.i.	Ingested by adults
	<i>Cryptolaemus montrouzieri</i>	Entobakterin®	1.0 a.i.	Ingested by adults

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
<i>S. cerealella</i>	*	Lab.	Kiselek, 1975	—	—
<i>C. pomonella</i>	****	Lab.	Kazakova and Dzhunosov, 1977	—	—
—	*	Lab.	Korostel and Kapustina, 1975	REP	24 h

—	****	Lab.	Tolstova and Ionova, 1976	—	24 h
<i>Hylemya brassicae</i>	*	Fld.	Obadofin and Finlayson, 1977	—	24—168 h
	*	Lab.			
	**	Lab.			
<i>Heliothis armigera</i>	*	Fld.	Umarov et al., 1975	—	1—360 h
	*				
<i>C. pomonella</i>	****	Lab.	Kazakova and Dzhunusov, 1977	—	—
	*				
<i>Aphis</i> sp.	**	Lab.	Kiselek, 1975	—	—

	*				
<i>P. brassicae</i>	*	Lab.	Legotai, 1980	—	—
<i>H. armigera</i>	*	Fld.	Umarov et al., 1975	—	1—360 h
					—
<i>Aphis punicae</i>	*	Lab.	Salama et al., 1982	REP, RHC	—
<i>H. armigera</i>	*	Fld.	Umarov et al., 1975	—	1—360 h
				—	1—360 h
	*	Lab.	Kiselek, 1975		

TABLE II (continued)

Non-target insect		Bacteria pathogen	Bacteria dosage	Method of exposure
Family	Species			
	<i>Hippodamia convergens</i>	Thuricide HPC®	Recommended field rate	Contact by adults
	<i>Stethorus loxoni</i>	<i>B. t.</i>	Recommended field rate	Contact by adults
	<i>S. nigripes</i>	<i>B. t.</i>	Recommended field rate	Contact by adults
		<i>B. t.</i>	0.375 g l ⁻¹ of 85% WP	Contact by adults
		<i>B. t.</i>	Recommended field rate	Contact by adults
Staphylinidae	<i>Paederus alfieri</i>	<i>B. t. var entomocidus</i>	500 µg ml ⁻¹	Ingested by adults
Chrysopidae	<i>Chrysopa carnea</i>	<i>B. t. var galleriae</i>	—	Ingested by larvae
		<i>B. t. var kurstaki</i>	—	Ingested by adults
		<i>B. t. var thuringiensis</i>	—	
		Dipel®	0.10% a.i.	Contact by adults
		Dipel®	1.0% a.i.	Ingested by larvae
		Entobakterin®	1.0% a.i.	
		Exotoxin®	1.0% a.i.	
		Toxobakterin®	1.0% a.i.	
		BTB-202®	0.5, 1.0, 1.5% a.i.	Ingested by larvae and adults
		<i>B. t. var entomocidus</i>	500 µg ml ⁻¹	Ingested by larvae
		Dendrobacillin®	2.0% a.i.	Contact and ingested
		Thuricide HPC®	Recommended field rates	Contact by adults and larvae
Anthrocoridae	<i>Orius niger</i>	Dendrobacillin®	2.0% a.i.	Contact and ingested
		Entobakterin®	2.0% a.i.	
Berytidae	<i>Jalysus spinosus</i>	Dipel®	16000 IU mg ⁻¹ 2 mg ml ⁻¹	Contact by adults
	<i>Picromerus bidens</i>	<i>B. t. var kurstaki</i>	1 × 10 ⁸ spores ml ⁻¹ 5 × 10 ⁸ spores ml ⁻¹	Ingested by adults

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
—	*	Lab.	Wilkinson et al., 1975	—	1-120 h
—	*	Lab.	Walters, 1976	—	—
—	*	Lab.	Walters, 1976	—	—
—	*	Lab.	Edwards and Hodgson, 1973	—	—
—	*	Lab.	Walters, 1976	—	1 and 18 h
<i>S. littoralis</i>	*	Lab.	Salama and Zaki, 1983	—	—
—	**	Lab.	Babrikova et al., 1982	—	—
—	***	Lab.		—	—
—	*	Lab.	Franz et al., 1980	—	—
<i>S. cerealella</i> eggs	**	Lab.	Kiselek, 1975	—	—
	*				
—	*	Lab.	Legotai, 1980	—	—
<i>S. littoralis</i>	*	Lab.	Salama et al., 1982	RHC	—
<i>H. armigera</i>	*	Fld.	Umarov et al., 1975	—	1-360 h
—	*	Lab.	Wilkinson et al., 1975	—	1-360 h
<i>H. armigera</i>	*	Fld.	Umarov et al., 1975	—	1-360 h
				—	1-360 h
<i>H. virescens</i>	*	Lab.	Dunbar and Johnson, 1975	—	48-480 h
<i>Y. evonymellus</i>	*	Lab.	Hamed, 1979	—	—

TABLE II (continued)

Non-target insect		Bacteria pathogen	Bacteria dosage	Method of exposure
Family	Species			
Miridae	<i>Campylomma diversicornis</i>	Dendrobacillin®	2.0% a.i.	Contact and ingested by adults
		Entobacillin®	2.0% a.i.	
	<i>Campylomma verbasci</i>	Dendrobacillin®	2.0% a.i.	Contact and ingested by adults
		Entobakterin®	2.0% a.i.	
Syrphidae	<i>Sphaerophoria scripta</i>	Bitoxi-bacillin 202®	70 milliard g ⁻¹	—
Tachinidae	<i>Bessa fugax</i>	<i>B. t. var kurstaki</i>	1×10^8 spores ml ⁻¹	Ingested by adults
	<i>Blepharipa pratensis</i>	Dipel 4L®	19.8 BIU ha ⁻¹	
	<i>Compsilura concinnata</i>	Dipel 4L®	19.8 BIU ha ⁻¹	
	<i>Pales pavidus</i>	Dipel®	0.10% a.i.	Contact by adults
	<i>Parasetigena silvestris</i>	Dipel 4L®	19.8 BIU ha ⁻¹	
	<i>Voria ruralis</i>	Thuricide HPC®	Recommended field rates	Contact by adults
	<i>Zenillia dolosa</i>	<i>B. t. var kurstaki</i>	1×10^8 spores ml ⁻¹	
			5×10^8 spores ml ⁻¹	
				Ingested by adults
Aeolothripidae	<i>Aeolothrips intermedius</i>	Dendrobacillin®	2.0% a.i.	Contact and ingested by adults
		Entobakterin®	2.0% a.i.	
Thripidae	<i>Scolothrips acariphagus</i>	Dendrobacillin®	2.0% a.i.	Contact and ingested by adults
		Entobakterin®	2.0% a.i.	
Mantidae	<i>Tenodera aridifolia sinensis</i>	<i>B. t. var kurstaki</i>	18,000 IU mg ⁻¹ 150 µg ml ⁻¹	Ingested by adults
Lycosidae	<i>Pardosa agrestis</i>	BTB-202®	0.5, 1.0, 1.5% a.i.	Ingested by adults

* = (0–10% mortality) no or little toxicity.

** = (10–40% mortality) low toxicity.

*** = (40–80% mortality) moderate toxicity.

**** = (80–100% mortality) high toxicity.

† REP = reduced egg production; RHC = reduced host consumption; RPP = reduced percent parasitism; RAL = reduced adult longevity; IPP = increased percent parasitism; IDT = increased developmental time; SSR = skewed sex ratios.

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
<i>H. armigera</i>	*	Fld.	Umarov et al., 1975	—	1-360 h
<i>H. armigera</i>	*	Fld.	Umarov et al., 1975	—	1-360 h 1-360 h
<i>C. pomonella</i>	****	Lab.	Kazakova and Dzhunosov, 1977	—	—
<i>Y. evonymellus</i>	*	Lab.	Hamed, 1979	—	—
<i>L. dispar</i>	**	Fld.	Ticehurst et al., 1982	RPP	—
<i>L. dispar</i>	**	Fld.	Ticehurst et al., 1982	RPP	—
—	*	Lab.	Franz et al., 1980	—	—
<i>L. dispar</i>	*	Fld.	Ticehurst et al., 1982	—	—
—	*	Lab.	Wilkinson et al., 1975	—	1-120 h
<i>Y. evonymellus</i>	*	Lab.	Hamed, 1979	—	—
<i>H. armigera</i>	*	Lab.	Umarov et al., 1975	—	1-360 h 1-360 h
<i>H. armigera</i>	*	Lab.	Umarov et al., 1975	—	1-360 h 1-360 h
<i>Trichoplusia ni</i>	*	Lab.	Yousten, 1973	—	168 h
<i>P. brassicae</i>	*	Lab.	Legotal, 1980	—	—

Formulation

Characterization of the different commercial formulations and strains of *B. t.* and *B. p.* may be important for three reasons: (1) different formulations have different carriers, ultraviolet screens and inert ingredients which may be toxic to non-targets; (2) different varieties of *B. t.* are formulated with different concentrations of active ingredients; (3) these bacterial varieties also may have different production rates of β -exotoxin and δ -endotoxin.

Few studies have analyzed the effects of the carrier substances, ultraviolet screening agents and inert ingredients used in commercial preparations of *B. t.* on non-target organisms (Table II). Haverty (1982) tested the effect of the non-aqueous oil carrier of Dipel 4L on all life stages of *Aphytis melinus*, *Hippodamia convergens* and *Chrysopa carnea* and found that mortality never exceeded 13.4%, even at an application rate of 18.71 ha^{-1} . Marchal-Segault (1975a) tested the spores, crystals and bentonite carrier of Bactospeine® separately after noting that the adult life spans of the parasitoids *Apanteles glomeratus* and *Phanerotoma flavitestacea* were significantly reduced by contact or consumption of the commercial formulation. The abrasive bentonite had a markedly adverse effect on the adult parasitoid's life span. Further testing is needed to determine the effect of inert ingredients and carriers on non-target insects.

Different commercial formulations of *B. t.* have different numbers of viable spores gram^{-1} (Forsberg et al., 1976). Concentrations range from 70 billion spores gram^{-1} for Agritol® to 2.5 billion spores gram^{-1} for Biotrol BTB 2.5D® (Forsberg et al., 1976). Formulations of Thuricide range from 3 billion spores gram^{-1} in Thuricide® to 65 billion spores gram^{-1} in Thuricide 65B® (Forsberg et al., 1976). Dulmage et al., (1976) detail a standardized procedure for *B. t.* that quantifies the problem of formulation variation appreciably. Often in the literature, information on commercial preparation and formulation is incomplete or lacking altogether.

The variety of subspecies of *B. t.* is very important because the serotypes and entomopathogenic components can be different. Varieties containing the heat stable -exotoxin may be the most detrimental in causing direct mortality of many beneficial insects (Tanada, 1984). Varieties of *B. t.* which are known to produce the -exotoxin include *thuringiensis*, *dendrolimus*, *morisoni*, *tolworthi* and *darmstadiensis*. Cantwell et al. (1972) report that Kreig and Herfs found a significant difference between five varieties of *B. t.* on honey bees when tested at a dose of 1×10^8 spores ml^{-1} . Three varieties had little effect, but *thuringiensis* and *dendrolimus* killed 100 and 60% of the bees, respectively, in nine days. The differences in mortality may reflect the differences in amount of exotoxin production by each strain (Forsberg et al., 1976). Formulations of *B. t.* that contain the β -exotoxin had a greater effect on mortality of *Coccinella septempunctata* larvae than formulations that did not contain the -exotoxin (Kiselek, 1975).

The actual dosage of pathogen applied to or ingested by a natural enemy

is often not clearly stated. With the exception of the work of Dulmage et al. (1976), no attempt has been made in the literature to standardize the dosage applications into international units as recommended by the 1966 symposium in Wageningen, The Netherlands (Forsberg et al., 1976). This makes it difficult to compare or contrast different tests using different formulations and different dosage measurements.

Testing protocol

As with viruses, an appreciable number (36% of those cited) of non-target arthropods tested in laboratory experiments (Table II) were exposed to bacterial pathogens by direct contact (e.g. sprayed, dipped or placed on residue). Of the 25 species tested by direct or residue exposure, only three showed greater than 10% mortality. Intuitively, these results seem straightforward because *B. t.*, usually, must be ingested by an insect to show a toxic effect. However, Tolstova and Ionova (1976) reported high mortality (100%) of adult *Trichogramma* sp. within 24 h when exposed to dried deposits of Dendrobacillin®. It has also been reported that the carabid *Bembidion lampros* incurred moderate mortality (15%) after 72 h of residual exposure to Dipel® (Obadofin and Finlayson, 1977).

Thus, it might be assumed that if significant mortality does occur to predators and parasitoids from direct contact with *B. t.*, then the lethal response may have occurred because the insects were adversely affected by an inert ingredient within the formulation or "internally contaminated" during the direct exposure testing. Internal contamination may occur if spores are (1) ingested while the insect is grooming, (2) consumed in a contaminated food source or (3) directly penetrate the body cavity through an integumental wound. It is highly unlikely that the spores, crystals or exotoxins of *Bacillus thuringiensis* would cause significant mortality of natural enemies when applied directly to their intact integument.

The majority of the bioassays done with *Bacillus thuringiensis* on non-target insects, test for acute pathological effects after spores have been ingested by the insects (Table II). For most predators and adult parasitoids, these tests were conducted by contaminating the prey or host with a known amount of the pathogen and monitoring uptake. The effect on parasitoid larvae was generally measured by allowing female parasitoids to oviposit in contaminated hosts or by feeding parasitized hosts a contaminated food source. Quantifying the effect on parasitoid larvae presents a special problem. As with the viruses, lethal and sub-lethal effects may not be directly induced by the bacteria, but may be indirectly caused by altering the available food source of the parasitoid larva. Marchal-Segault (1975b,c) showed significant indirect mortality of the larvae of two braconid parasitoids when their host died before the parasitoid completed its development. The main effect was to lower the food quality rather than poison the host-feeding larvae (Marchal-Segault, 1975b). A high percentage of indirect mortality was also

reported in *Microplitis demolitor* when *Spodoptera littoralis* died before *M. demolitor* completed its development (Salama et al., 1982). In much of the literature, it is never stated whether reduction in parasitoid emergence from *B. t.* infected hosts is due to direct or indirect mortality (Table II).

Quantifying the amount of bacteria ingested by the parasitoid larvae is also difficult. Some parasitoid larvae do not consume their entire host and only feed upon a specific organ or group of organs (e.g. the fat body of lepidopterous larvae). Thus, a parasitoid could possibly complete development within an infected host and never ingest toxins within the host tissue or haemolymph.

The particular life stage of the organism being tested may also affect the results of the bioassay (Table II). Considerable literature has been published which presents observations or discussions of the factors which seem to confer a greater chemical pesticide tolerance for a given life stage of a natural enemy as compared to another. In general, it is the adult stage of the parasites and predators which are considered to be the most susceptible to chemical insecticides, although a few species of larval coccinellids are an exception to this generalization (Croft, 1977). It appears that the opposite may be true when parasitoids and predators are exposed to high dosages of *B. t.* In general, it is the larvae of natural enemies that are more susceptible to *B. t.* than the adults (Tanada, 1984), but there is little quantitative data to support this.

Kiselek (1975) found that Dipel® and Exotoxin® caused 20 and 18% mortality of *Chrysopa carnea* larvae, respectively, but were harmless to the adults. Conversely, Babrikova et al. (1982) found that three varieties of *B. t.* caused 75% mortality in adult *C. carnea*, but only 30% in the larvae. Kiselek (1975) also reported that 28 and 90% of *Coccinella septempunctata* larvae were killed by Entobakterin® and Exotoxin®, respectively, yet neither formulation had an effect on the adults. Kazakova and Dzhunusov (1977) reported 100% mortality of adult *Trichogramma* sp., but no mortality if larvae were exposed to Bitoxibacillin 202®. The effects of other types of bacteria on different life stages have been investigated even less. Bracken and Bucher (1967) found that the bacterial pathogen *Serratia marcescens* was highly toxic to the adult ichneumonid *Exeristes comstockii*, yet had no noticeable effect on the ichneumonid larva. In this situation, the larvae of the parasitoid were contaminated while feeding upon their infected host, *Galleria mellonella*, but mortality did not occur until the adult stage. Survival of the larvae was normal (over 90%) suggesting that parasitoid larvae could tolerate considerable ingestion of *S. marcescens*; but the bacteria remained within the parasitoids causing 100% mortality of adults due to septicemia (Bracken and Bucher, 1967).

One additional explanation for the variability of results between different bioassays, testing similar varieties of *B. t.* on the same organisms (Table II), is the amount of time allotted to incubate a lethal response. This time is often the same as those standardly used in chemical dose/time response

tests (i.e. 24, 48 and 72 h). However, many tests have shown that direct lethal effects of *B. t.* on predators and parasitoids increase, when assayed over a longer period of time. For example, the LD₅₀ of Thuricide HP® for honey bee larvae is 9×10^8 spores g⁻¹ if measured after 48 h and only 0.2×10^8 spores g⁻¹ if measured at 96 h (Ali et al., 1973). Thus, a reduction in dosage of over 95% gives the same lethal dose response when measured after a longer growth period. The phenomenon is modified if the predator or parasite must first consume its host(s) before it ingests a bacterial pathogen. Therefore, it might be advisable to lengthen lethal dose/time response measurements in bioassays for those agents to allow for pathogen reproduction and infection. Obviously, duration of assays will be limited by the length of time that an insect can persist under the stated test conditions.

Lethal effects

Both direct and indirect lethal effects of bacterial pesticides to non-target, beneficial insects have been observed with various formulations of *B. t.* (Table II). Direct mortality of parasitoids from infection and septicemia has been documented by Hamed (1979), Muck et al. (1981), Thompson et al. (1977) and Niwa (1985). Hamed tested 7 species of adult parasitoids and one predator of *Yponomeuta evonymellus*. He found that 2 species of tachinids, *Bessa fugax* and *Zenillia dolosa*, as well as one ichneumonid, *Agrypon* sp. and the hemipteran *Picromerus bidens* were not susceptible to *B. t.* var. *kurstaki*. However, 2 ichneumonids, *Diadegma armillata* and *Pimpla turionellae*, 1 encyrtid, *Ageniaspis fuscicollis*, and the eulophid *Tetrastichus evonymellae* were found to be susceptible after ingesting spores of *B. t.* with food sap. Large numbers of vegetative cells of *B. t.* were detected in the haemolymph and gut of the dead parasites.

Muck et al. (1981) also tested the ichneumonid parasitoid *Pimpla turionellae* and the braconid parasitoid *Cotesia glomeratus* with *B. t.* var. *kurstaki* (Dipel®). They found that high concentrations applied orally (e.g. 10^8 and 10^9 spores ml⁻¹) significantly increased the mortality of *C. glomeratus* after 2 weeks (39 and 100% mortality, respectively, compared to 9% in the control). *Pimpla turionellae* was less affected than *C. glomeratus*. Eighteen days after the continuous application of 10^7 , 10^8 and 10^9 spores ml⁻¹, *P. turionellae* mortality reached 13, 18 and 26%, respectively, compared to 2% in the control. Histopathological examination of *P. turionellae* after ingestion of Dipel® at the rate of 10^9 spores ml⁻¹ indicated that damage to the mid-gut was caused by the endotoxin of *B. t.*

Direct mortality of parasitoids has been observed by Thompson et al. (1977) in field tests using *B. t.* against the western spruce budworm, *Choristoneura occidentalis* (and the douglas fir tussock moth, *Orgyi a pseudotsugata*). They found that two important budworm parasitoids, *Glypta fumiferanae* and *Apanteles fumiferanae*, could acquire lethal *B. t.* infections from their budworm host. Fully developed parasite larvae would

emerge from their host only to die before they could pupate. Upon inspection, these dead parasites were found to contain all stages of the bacillus and were presumed to have died from infection. In almost all cases the host budworm remained alive until after the parasitoid emerged.

Predators may also be directly affected by *B. t.* Direct mortality of *Coccinella septempunctata* has been reported by Kazakova and Dzhunusov (1977) and Kiselek (1975), using Bitoxibacillin®, Entobakterin® and Exotoxin®, however, other bacillus formulations tested against *C. septempunctata* and other coccinellids have had little or no effect (Table II). Similar results have been obtained for the lacewing predator *Chrysopa carnea*. Babrikova et al. (1982) and Kiselek (1975) have measured significant mortality in lacewings when the larvae and adults were fed several varieties of *B. t.* (Table II). Legotai (1980), Salama and Zaki (1983) and Umarov et al. (1975) found that feeding *C. carnea* other formulations and varieties of *B. t.* had little or no measurable effect.

Sub-lethal effects

Effects from consumption of sub-lethal dosages of bacillus or consumption of sub-lethally infected hosts have been reported for several predators and parasitoids (Table II). Documented sub-lethal effects include: reduced egg production, reduced host consumption, reduced percent parasitism, increased percent parasitism, reduced adult longevity, increased parasitoid development and skewed sex ratios. Often it is not apparent whether these effects are caused by direct (e.g. sub-lethal response of the natural enemy) or indirect factors (e.g. sub-lethal response of the host rendering it more or less suitable as an available food source).

Total egg production was reduced by 27% when the braconid parasitoid *Microplitis demolitor* and the ichneumonid parasitoid *Zele chlorophthalma* were reared on *Spodoptera littoralis* infected with *B. t.* var. *entomocidus* (Salama et al., 1982; Salama and Zaki, 1983). Egg production by the braconid *Bracon brevicornis* was reduced by 36 and 69% when it was reared on *Sesamia cretica* injected with 3×10^{10} and 3×10^{16} spores ml^{-1} of *B. t.*, respectively (Temerak, 1980). The predator *Coccinella undecimpunctata* produced 30% fewer eggs after being fed aphids sprayed with a $500 \mu\text{g ml}^{-1}$ suspension of *B. t.* (Salama et al., 1982). Korostel and Kapustina (1975) reported that adult *Trichogramma* sp. that survived consumption of *B. t.* had reduced fecundity.

Total host consumption rate for the predator *Chrysopa carnea* was reduced by 34 and 26%, respectively when fed infected *S. littoralis* and contaminated *Aphis durante* (Salama et al., 1982). Consumption of aphids by *C. undecimpunctata* was reduced by 30% when *B. t.*-contaminated *A. durante* were fed to the coccinellid larvae (Salama et al., 1982).

Percent parasitism of *S. littoralis* by the ichneumonid parasitoid, *Zele*

chlorophthalma, was reduced by 75% compared to the control when the host larvae were fed on a diet containing *B. t.* Decrease in percent parasitism has also been reported for two tachinid parasites of the gypsy moth, *Comp-silura concinnata* and *Blepharipa pratensis* in field plots sprayed with *B. t.* (Ticehurst et al., 1982).

Surprisingly, field studies also show that in plots treated with *B. t.* percent parasitism of host species is often greater than in untreated plots. Parasitism of the gypsy moth, *Lymantria dispar* larvae by *Cotesia melanoscelus* and density of the parasitoid's cocoons were greater in hardwood plots treated with *B. t.* than in untreated plots (Ticehurst et al., 1982; Weseloh et al., 1983). These field plots showed a strong correlation between percent parasitism and gypsy moth larval size. *C. melanoscelus* parasitizes the small early instars of *L. dispar*. Gypsy moth larvae that survive *B. t.* doses have retarded developmental rates (Weseloh and Andreadis, 1982; Weseloh, 1984). Thus, the retarding effect of *B. t.* infection kept *L. dispar* larvae smaller for a longer period of time than untreated larvae and this permitted females of *C. melanoscelus* to parasitize larger numbers of hosts (Weseloh et al., 1983).

This same phenomenon has been shown to occur for the introduced braconid parasitoid of the gypsy moth *Rogas lymantriae* (Wallner et al., 1983). In addition, the sex ratio of *R. lymantriae* was significantly skewed in *B. t.*-treated plots because the female parasitoid oviposited a higher percentage of male progeny in the smaller hosts (Wallner et al., 1983).

Rate of parasitoid development was shown to be affected when parasitoids were reared on infected hosts. When *Ephesia kuehniella* and *Pieris brassicae*, the respective hosts of the parasites *Phanerotoma flavitestacea* and *Cotesia glomeratus*, were fed sub-lethal doses of Bactospeine®, it caused a reduction in the normal food consumption of both host species and consequent lengthening of the larval period for both the hosts and their parasitoids (Marchal-Segault, 1975b). Ahmad et al. (1978) found that treating gypsy moth larvae with Dipel HG® caused an increase of approximately 3 days in the total development time of its braconid parasitoid *C. melanoscelus*. Similar results were obtained when *Zele chlorophthalma* parasitized bacillus-infected *Spodoptera littoralis* (Salama and Zaki, 1983).

Adult longevity has been shown to be adversely affected when adult parasitoids were exposed to or ingested *B. t.* (Table II). The longevity of adult *P. flavitestacea* was reduced from 70 days for no treatment to 34 and 20 days when the adult parasitoid was infected by contact with *B. t.* used at 1 and 5% active ingredient (a.i.), respectively (Marchal-Segault, 1975a). Reduction of adult longevity for *C. glomeratus* when ingesting a sugar solution containing 1 and 5% a.i. of *B. t.* was from 24 days for the control to 13 and 10 days in the respective treatments (Marchal-Segault, 1975a). Adult *Bracon brevicornis* also had significantly shorter life-spans when they were kept in vials with host larvae which had been infected with *B. t.* (Temerak, 1980).

Ecological relationships and selectivity

Often the physiological or ecological characteristics of an organism allow it to escape a potentially toxic pesticide. If an organism is in a less susceptible or protected life stage (e.g. diapause, quiescence or refuge) or is not residing in the habitat during application of the toxin, then the impact on that organism in the field may be less than predicted from laboratory experiments (ecological selectivity). *B. t.* exhibits relatively high levels of selectivity for many natural enemies due to the specific physiological mechanisms required to initiate infection. Ingestion of spores by adult predators and parasites could occur only if the insect (1) consumes the bacillus by host feeding, (2) ingests spores while grooming or cleaning sensory organs that were contaminated with the bacteria or (3) ingests spores while nectar feeding. Thus, the residual toxicity of *B. t.* to adult predators and parasitoids with an intact integument may be of little real ecological significance.

Most predacious Hemiptera and Coleoptera are insectivorous both as larvae and adults. However, some of the important predators, such as syrphids, chrysopids, the majority of the parasitic diptera and many parasitic hymenoptera do not host feed as adults. The question then becomes, how many viable spores do adult insects contact during their daily activity within a habitat sprayed with *B. t.*? At the recommended field rate, the average number of bacillus spores m^{-2} is 11×10^9 spores m^{-2} (1.1×10^4 spores mm^{-2}) (Forsberg et al., 1976). This field rate is a lower concentration ($0.001 \times$) than most of those used to initiate high mortality of adult parasitoids in the laboratory by contamination of food sap (e.g. 1×10^9 spores $ml^{-1} \approx 10^7$ spores mm^{-2})¹ (Muck et al., 1981). Thus, the probability that an adult parasitoid would consume lethal doses of *B. t.* while searching for hosts and nectar feeding within *B. t.*-sprayed field might be very low, however, this has not been investigated. The amount of *B. t.* ingested by host-feeding parasitoids, likewise, has not been investigated. The likelihood of lethal *B. t.* consumption becomes even more remote if the parasitoids' nectar feeding is restricted to an area outside the habitat sprayed with *B. t.*

Parasitoid larvae may also escape exposure to *B. t.* They may alter the behavior of their host making it less likely to consume the bacillus. For example, Hamel (1977) found that two parasitoids of the western spruce budworm, *Choristoneura occidentalis*, increased in Dipel®-treated field plots over a 21-day period compared to untreated plots. Three other parasitoid species decreased over the same time period. *Apanteles fumiferanae* and *Glypta fumiferanae* both attack pre-diapausing 1st instar budworm larvae. The parasitized budworm larvae were negatively phototrophic, which reduced their feeding stimulus in the spring when the bacillus was applied. Most other parasitoids of the budworm attack later instars which contacted

¹ Estimated spore density on surface assuming removal of all liquid.

and ingested, the bacillus (Hamel, 1977). Thus, even if *A. fumiferanae* and *G. fumiferanae* were shown to be susceptible to *B. t.* in the laboratory, the probability of field infection may be reduced. Marchal-Segault (1975c) has also shown that the presence of the parasites *Phanerotoma flavitestacea* and *Cotesia glomeratus* within their lepidopterous host *Pieris brassicae* and *Ephestia kuehniella*, respectively, actually delayed or hindered the rate of the lepidopteran's bacterial infection because their feeding rates on microbial-sprayed food was reduced. Thus, parasitized larvae may have a higher probability of avoiding or delaying a lethal bacillus dosage than unparasitized larvae.

A mechanism by which adult parasitoids can avoid direct or indirect mortality of their progeny from a pathogen is by being able to discriminate between infected and uninfected hosts. It has been shown that *C. melanoscelus* females can discriminate between non-infected and NPV-infected gypsy moth larvae (Versoi and Yendol, 1982). This may also be the case with *B. t.*-infected larvae. The ichneumonid *Zele chlorophthalma* parasitized 79% of *Spodoptera littoralis* fed on untreated artificial diet, but only 20% of the *S. littoralis* fed on diet treated with *B. t.* (Salama and Zaki, 1983). These results suggest that *Zele chlorophthalma* finds infected *S. littoralis* less suitable than uninfected hosts. The extent to which hymenopterous parasitoids can discriminate between *B. t.*-infected and uninfected hosts has not been directly investigated.

FUNGI

Entomogenous fungi are widely distributed and are an important factor in natural insect control worldwide (Cherwonogrodzky, 1980). All four taxonomic groups of the fungi contain entomopathogenic species. The majority of publications on insect mycoses discuss members of the genera *Beauveria*, *Metarrhizium* and *Aspergillus* (Burges and Hussey, 1971). Species most commonly used in biological control programs are members of the Fungi Imperfecti and include *Beauveria bassiana*, *Metarrhizium anisopliae*, *Nomuraea rileyi* and *Paecilomyces farinosus*. *Beauveria bassiana* is produced commercially under the trade name Boverin®.

Mode of infection is generally by cuticular penetration by the germ tube of the fungal conidia, although infection may occasionally occur through the respiratory or alimentary tract (Burges and Hussey, 1971). After germination, the mycelium penetrates the tissue by the release of enzymes and mechanical pressure (Cherwonogrodzky, 1980). Free floating "yeast-like" mycelial fragments are produced which multiply in the hemocoel. Death of the host can occur either from the release of fungal toxins or by tissue destruction. Hyphal growth continues until the insect is filled with mycelia, at which point fruiting bodies erupt through the cuticle, spreading spores to other susceptible insects (Cherwonogrodzky, 1980).

TABLE III

Effects of fungal pathogens on non-target insects

Non-target insect		Fungus pathogen	Fungus dosage	Method of exposure
Family	Species			
Aphelinidae	<i>Encarsia formosa</i>	Boverin® (<i>Beauveria bassiana</i>)	recommended rate	Contact by adults
		<i>Verticillium lecanii</i>	3.6×10^7 blastospores ml^{-1}	Contact by adults
Apidae	<i>Apis mellifera</i>	<i>Hirsutella thompsonii</i>	271 CFU	Ingested by adults
Braconidae	<i>Cotesia glomeratus</i>	<i>Beauveria bassiana</i>	—	Infects host after female oviposits
	<i>Microplitis croceipes</i>	<i>Nomuraea rileyi</i>	1×10^9 spores larvae^{-1}	Brushed on bollworm
Trichogrammatidae	<i>Trichogramma cacoeciae</i>	Boverin®	0.5% a.i.	Contact by adults
	<i>T. embryophagum</i>			
	<i>T. evanescens</i>			
Carabidae	<i>T. pallidum</i>	Boverin®	1% a.i.	Sprayed on host eggs
	<i>Brosicus cephalotes</i>	<i>Beauveria bassiana</i>	—	Contact by adults
	<i>Harpalus rufipes</i>	<i>Paecilomyces farinosus</i>	—	
	<i>Pterostichus vulgaris</i>	<i>Paecilomyces fumosoroeus</i>	—	
Coccinellidae	<i>Coccinella septempunctata</i>	<i>Beauveria bassiana</i>	1%	Ingested by adults and larvae
	<i>Cryptolaemus montrouzieri</i>	Boverin®	1%	Ingested by adults and larvae

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
<i>Trialeurodes vaporariorum</i>	*	Lab.	Beglyarov and Maslenco, 1978	—	240 h
<i>T. vaporariorum</i>	*	Lab.	Hall, 1982	—	—
—	**	Lab.	Cantwell and Lehnert, 1979	—	—
<i>Pieris brassicae</i>	*	Lab.	Fuhrer and El-Sufy, 1979	—	—
<i>Heliothis zea</i>	*	Lab.	King and Bell, 1978	—	—
—	*	Fld.	Kapustina, 1977	—	—
				—	—
	*	Lab.	Kiselek, 1975	—	—
—	*	Lab.	Kabacik-Wasylik and Kmitowa, 1973		—
	*	Fld.	Kmitowa and Kabacik-Wasylik 1971	IOM	
	*				
—	*	Lab.	Kiselek, 1975	—	—
	*				
<i>Sitotroga cerealella</i>	*** (larvae)	Lab.	Kiselek, 1975	—	—

TABLE III (continued)

Non-target insect		Fungus pathogen	Fungus dosage	Method of exposure
Family	Species			
Chrysopidae	<i>Chrysopa carnea</i>	<i>Beauveria bassiana</i>	—	Sprayed on fields
		Boverin®	1%	Ingested by adults and larvae
Pentatomidae	<i>Perillodes bioculatus</i>	<i>Beauveria bassiana</i>	—	Sprayed on field

* = (0–10% mortality) no or slight toxicity.

** = (10–40% mortality) low toxicity.

*** = (40–80% mortality) moderate toxicity.

**** = (80–100% mortality) high toxicity.

†IOM = increased overwintering mortality.

The fungi are highly dependent on the microclimate surrounding their host. The limits of fungal growth range between 5 and 35°C with the optima between 20 and 30°C. High humidity is critical to germinate spores and to increase spore production on fruiting bodies. Light has also been shown to affect sporulation on the host after it is dead as well as longevity of spore viability (Roberts and Yendol in Burges and Hussey, 1971).

Table III lists the effects of assays conducted with entomogenous fungi on non-target beneficial insects. The majority of this research has been done in the U.S.S.R. and often only English abstracts were available for interpretation of the data. This table does not include any information on the effect of *Culicinomyces* sp. on non-target aquatic insects. For a review of this literature, see Knight (1980).

Formulation

Currently, fungal pathogens are not being pursued as commercial products in the U.S.A. (Cherwonogrodzky, 1980). *Beauveria bassiana* is mass produced in a factory at Krasnodar in the U.S.S.R. under the registered name of Boverin®. In China, *B. bassiana* is also produced and widely used for insect control. Although some of the entomogenous fungi are host specific, others, such as *B. bassiana*, have a very broad host range. *B. bassiana* is probably not mass produced commercially in the U.S.A. due to inconsistencies and unreliability in reducing pest populations below threshold levels during field trials. However, as research continues abroad, demand for alternatives to chemical control increase, and human allergic responses to this microbial are better evaluated, we might see this product manufactured in the U.S.A.

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
<i>Leptinotarsa decemlineata</i>	*	Fld.	Gusev et al., 1975	—	—
<i>S. cerealella</i>	*	Lab.	Kiselek, 1975	—	—
<i>L. decemlineata</i>	*	Fld.	Gusev et al., 1975	—	—

Testing protocol

The majority of non-target insects tested with fungal pathogens were inoculated by allowing the insect to contact the entomogenous fungal spores directly (Table III). Toxicity was low and mortality never exceeded 10%. Information on the micro-environment surrounding the host was usually lacking, so it is not known whether these fungi were tested under optimal or sub-optimal conditions for their growth and population increase. Surprisingly, the only tests to show greater than 10% mortality were those in which the natural enemies consumed the fungal spores. Ingestion of fungal spores by natural enemies could be important if large areas were to be sprayed with entomogenous fungi.

Lethal effects

Kiselek (1975) reported that 50% of the coccinellid larvae *Cryptolaemus montrouzieri* died when they consumed mealybugs sprayed with a 1% a.i. solution of Boverin®. Boverin® was harmless to *Cryptolaemus* adults. Cantwell and Lehnert (1979) reported 29% mortality of worker bees that were fed spores of *Hirsutella thompsonii*. All remaining tests reported no to very low mortality of natural enemies exposed to entomogenous fungi spores (Table III). Indirect mortality of *Microplitis croceipes* was reported to occur if its larval host, *Heliothis zea*, was predisposed to *Nomuraea rileyi* infection before oviposition, or if fungal infection occurred within 24 h of parasitization (King and Bell, 1978). If *Nomuraea rileyi* infected *H. zea* 24 h after parasitization, then the parasitoid could complete its development and emerge before the *Heliothis* died.

Sub-lethal effects

Few studies investigated the sub-lethal effects of entomogenous fungi on natural enemies and beneficial insects. Cantwell and Lehnert (1979) found no abnormalities in egg production, brood rearing, worker and queen mortality or general colony behavior when hives of *Apis mellifera* were fed *Hirsutella thompsonii*. Overwintering mortality of the carabid beetle *Harpalus rufipes* was slightly increased when cultures of the fungi *Paecilomyces farinosus* and *Paecilomyces fumosoroseus* were incorporated into soils in which the adults overwintered. In the same tests, *B. bassiana* did not increase overwintering mortality (Kabacik-Wasylik and Kmitowa, 1973).

Increased overwintering mortality could be an important lethal effect of many natural enemies if large quantities of entomogenous fungi were to be used commercially. In England, overwintering mortality of the coccinellid *Adalia bipunctata* was 36%, the primary cause of death was the fungal pathogen *Beauveria bassiana* (Mills, 1981). Hibernating adults of *Coccinella septempunctata* are also attacked by *B. bassiana* and infection rates of 50% have been reported in overwintering populations in southeastern France (Hodek, 1973). Thus, by increasing the amount of inoculum as a result of the application of an entomophagous fungal insecticide to crops, the natural overwintering mycosis of many inactive non-target insects might be increased, particularly if the fungal pathogen has a cosmopolitan host range.

Ecological relationships and selectivity

Despite the broad host range of many entomogenous fungi, the assays conducted on non-target beneficial insects using fungal pathogens have shown this group to be relatively selective (Table III). However, this statement is based on a small number of tests, many of which have not been completely translated into English. One interesting ecological relationship that was documented is that parasitism may predispose the host to fungal infection. King and Bell (1978) found that the rate of infection from the fungus *N. rileyi* on *Heliothis zea* was extremely low if applied before parasitization by *M. croceipes*. However, when the fungus was applied either at the same time or after the parasite attack, the infection rate was significantly higher, indicating that *Microplitis* predisposed the host to infection by the fungus. They also found that if the parasite and fungus were introduced into the host at the same time, then *Microplitis croceipes* required a significantly longer period of time (1.1 days) to complete development than was required for *N. rileyi* to kill *H. zea*. Therefore, indirect parasitoid mortality from the pathogen would occur (King and Bell, 1978).

Parasite-induced host susceptibility by the fungus *Beauveria bassiana* has also been reported for another braconid, *Cotesia glomeratus* (Fuhrer and El-Sufy, 1979). This parasitoid reduces fungal competition by production of a fungistatic substance that has been shown to inhibit the development of

B. bassiana in the haemolymph of *Pieris brassicae*. This substance was found to be produced in the teratocytes of the immature *C. glomeratus*. The derivatives of the parasite serosa float in the haemolymph producing a fungistatic material that compensates for the enhanced susceptibility of the host (Fuhrer and El-Sufy, 1979). Thus, this might suggest one possible mechanism evolved by braconid parasitoids to reduce fungal mycosis induced by oviposition.

PROTOZOA

Entomopathogenic protozoa are taxonomically diverse and include members of the flagellates, ciliates, amoebae, coccidia, haplosporida, neogregarines and microsporida (Burgess and Hussey, 1971). Many of the protozoa that show some commercial potential are microsporida, particularly members of the genus *Nosema*. The primary mode of infection is by ingestion; however, transovum and transovarial transmission have also been documented. Cuticular penetration is also known to occur in the Tetrahymenidae ciliates, a parasite of mosquitoes (Cherwonogrodzky, 1980). Protozoa are seldom lethal, but reduce insect fitness by lowering vitality, progeny number, life span and stimuli responses. Once thought to be highly host specific, most are now known to have cosmopolitan host ranges with the ability to cross-infect several orders of insects.

Table IV lists the effects of assays conducted with protozoa on non-target beneficial insects. The majority of these assays were conducted on hymenopterous parasitoids. This table does not include information on the effects of protozoa on non-target aquatic insects (for this information see Essen and Anthony, 1976).

Formulation

Currently, most protozoa are not being pursued as commercial insecticides. However, *N. locustae* is produced under the trade name Nolo® to reduce grassland grasshopper infestations. Most protozoa are not considered commercially attractive because the characteristics of the pathogens suggest that they cannot be used as short-term, quick-acting microbial insecticides (Burgess and Hussey, 1971). Economically they must be produced in vivo, so commercial manufacture would be labor intensive and costly. Also, few act with sufficient speed to prevent damage to crops, unlike other microbials such as viruses or *Bacillus thuringiensis*. If damage is to be circumvented using protozoa, the pathogens must be introduced in the season or developmental stage prior to an expected pest outbreak (Burgess and Hussey, 1971). Thus, as advances are made and integrated pest management and pest outbreak prediction improves, protozoa may have an increasing commercial potential.

TABLE IV

Effects of protozoa on non-target arthropods

Non-target insect		Protozoa pathogen	Protozoa dosage	Method of exposure
Family	Species			
Apidae	<i>Apis mellifera</i>	<i>Nosema locustae</i>	5×10^1 5×10^4 spores bee ⁻¹	Ingested by adults
Braconidae	<i>Ascogaster quadridentata</i>	<i>Nosema carpocapsae</i>	—	Ingested by larvae
	<i>Bracon mellitor</i>	<i>Glugea gasti</i>	100% infected hosts	Ingested by larvae
		1069 (unidentified)		
		<i>Mattesia grandis</i>	1×10^7 spores mm ⁻²	Ingested by larvae and adults
	<i>Cardiochiles nigricaps</i>	<i>Nosema campoletidis</i>	—	Contact and ingested by parasitoid larvae
		<i>Nosema heliothidis</i>	—	
	<i>Macrocentrus grandii</i>	<i>Nosema pyrausta</i>	—	Ingested by larvae
	<i>Cotesia glomeratus</i>	<i>Nosema polyvora</i>	—	Ingested by larvae
		<i>Nosema mesnili</i>	—	Ingested by larvae or direct penetration of larvae
Chalcidae	<i>Spilochalcis side</i>	<i>Nosema campoletidis</i>	—	Contact
		<i>Nosema heliothidis</i>	—	
Ichneumonidae	<i>Campoletis sonorensis</i>	<i>Nosema campoletidis</i>	—	Exposed to host larvae
		<i>Nosema heliothidis</i>	—	

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
Acrididae	*	Lab.	Menapace et al., 1978	—	624 h
<i>Cydia pomonella</i>	****	Lab.	Huger and Neuffer, 1978	—	—
<i>Anthonomus grandis</i>	****	Lab.	Bell and McGovern, 1975	—	1-360 h
	*			—	1-360 h
<i>A. grandis</i>	**	Lab.	McLaughlin and Adams, 1966	RPP	1-168 h
<i>Heliothis zea</i> <i>H. virescens</i>	**	Fld.	Brooks and Cranford, 1972	—	1-336 h
	*			—	1-336 h
<i>Ostrinia nubilalis</i>	**	Fld.	Andreadis, 1980, 1982	RAI	—
<i>P. brassicae</i>	***	Lab.	Iasi and Maslennikova, 1964	RNW, RF	—
<i>P. brassicae</i>	***	Lab.	Larson, 1979	—	1-2400 h
<i>Campoletis sonorensis</i>	*	Lab.	McNeil and Brooks, 1974	—	1-2400 h
	*			—	1-2400 h
				—	1-2400 h
<i>H. zea</i> <i>H. virescens</i>	*			—	1-240 h
	*			REP, RAE	—

TABLE IV (continued)

Effects of protozoa on non-target arthropods

Non-target insect		Protozoa pathogen	Protozoa dosage	Method of exposure
Family	Species			
Trichogrammatidae	<i>Trichogramma evanescens</i>	<i>Nosema pyrausta</i>		Ingested by larvae
Pteronallidae	<i>Catolaccus aeneo-viridis</i>	<i>N. campoletidis</i>	—	Contact
		<i>N. heliothidis</i>	—	
Reduviidae	<i>Zelus exsanguis</i>	<i>Varimorpha necatrix</i>	300 spores mm^{-1}	Ingested by saltmarsh caterpillar
		<i>Pleiotropha</i> sp.	500 spores mm^{-2}	

* = (0–10% mortality) no or slight toxicity.

** = (10–40% mortality) low toxicity.

*** = (40–80% mortality) moderate toxicity.

**** = (80–100% mortality) high toxicity.

† REP = reduced egg production; RPP = reduced percent parasitism; RAL = reduced adult longevity; RMW = reduced mean weight; RF = reduced fecundity.

Testing protocol

Over 80% of the insects tested were assayed by allowing the beneficial insect to ingest the protozoa (Table IV). Toxicity to non-target insects ranged from high (Bell and McGovern, 1975; Huger and Neuffer, 1978), to low (Kaya, 1979) depending on the protozoa and non-target organism tested. Tests for contact toxicity were very low, as would be expected, because the protozoan must enter the alimentary canal to have a toxic effect. Duration of tests were longer than most of the other microbial assays, with one test terminated after 100 days (McNeil and Brooks, 1974). Protozoan tests should be relatively longer than other assays because seldom are vital functions of the host disrupted quickly, rather a slow debilitation generally occurs (Burgess and Hussey, 1971).

Lethal effects

Mass rearing of a codling moth parasitoid, *Ascogaster quadridentata*, failed when *A. quadridentata* became infected with *Nosema carpocapsae*, a microsporidian parasite of the codling moth (Huger and Neuffer, 1978). Thus, the parasitoid colony crashed due to infection from a microsporidian of its host. A similar phenomenon occurred when Bell and McGovern (1975) attempted to rear *Bracon mellitor* on boll weevil they had purposely

Treated insect host	Toxicity	Test site	Reference	Sub-lethal effects	Duration
<i>O. nubilalis</i>	***	Lab.	Huger, 1984	—	—
<i>C. sonorensis</i>	*	Lab.	McNeil and Brooks, 1974	—	—
<i>Estigmea acrea</i>	*	Lab.	Kaya, 1979	—	—
	*				

infected with the microsporidian *Glugea gasti*. No parasites emerged from weevil larvae infected with the microsporidia. Immature *B. mellitor* dissected from infected hosts all contained large numbers of *G. gasti* spores, suggesting that mortality was directly induced by the protozoan (Bell and McGovern, 1975). *B. mellitor* was also found to be susceptible to a neogregarine, *Mattesia grandis*, when it ingested all stages of the protozoan while feeding in infected boll weevils (McLaughlin and Adams, 1966). Andreadis (1982) found a significant inverse correlation between the prevalence of infection of the European corn borer, *Ostrinia nubilalis*, with the microsporidian *Nosema pyraustae* and the prevalence of parasitism by the introduced braconid *Macrocentrus grandii* when infections with *N. pyraustae* exceeded 45% of the corn borer population. These findings strongly suggest that *N. pyraustae* had a significant adverse effect on field populations of *M. grandii* and may help explain the diminishing role of this and other introduced parasitoids as natural control agents of the European corn borer in the U.S.A. (Andreadis, 1982).

Sub-lethal effects

Many sub-lethal effects for parasitoids infected by a host protozoa have been documented; these include reduced adult longevity, reduced percent parasitism, reduced fecundity and reduced mean adult weight (Table IV).

Andreadis (1980) found that *Macrocentrus grandii*, which developed within larvae of *Ostrinia nubilalis* infected with *Nosema pyraustae*, had significantly shorter adult life-spans. The life-span of adult males was reduced from 14.3 days for uninfected to 2.3 days for infected individuals. The life-span of adult females was reduced from 16.6 days for uninfected to 1.9 days for infected individuals. *Nosema pyraustae* also adversely affected the pupal development of *Macrocentrus grandii*.

An egg parasitoid of *Ostrinia nubilalis*, *Trichogramma evanescens*, had its mean fecundity rate reduced by 50% when female *Trichogramma* were infected with *N. pyrausta*. Huger suggests that there might be normal parasitism rates when *T. evanescens* is initially released into the field. However, an incidence of *N. pyrausta* in *O. nubilalis* eggs might reduce the parasitizing capacity in the infected F_1 generation of the parasitoids (Huger, 1984). Reduced fecundity was also observed in *Cotesia glomeratus* that parasitized *Pieris brassicae* infected with *Nosema polyvora*. In addition, reduced mean adult weight was noted; the smaller specimens also suffered a greater proportion of overwintering mortality (Issi and Maslennikova, 1964). Percent parasitism was reduced by 40% when *Bracon mellitor* was infected with the protozoa *Mattesia grandis* from its host, the boll weevil (McLaughlin and Adams, 1966).

Ecological relationships and selectivity

Unlike the other microbials, most of the protozoan tested in these assays were capable of infecting the parasitoids of their hosts and subsequently having some effect on their fecundity or fitness (Table IV). This type of effect on natural enemies is not easily quantifiable, especially in assays of short duration, because indirect impact on population dynamics are often difficult to detect and may be overlooked for several generations. This effect is not usually caused by direct mortality, but rather by slowly reducing parasitoid fitness. For example, Issi and Maslennikova (1964) found that when *Cotesia glomeratus* was infected by *Nosema polyvora* transmitted from its host *Pieris brassicae*, the percent of individuals that went into diapause was greatly reduced. Thus, *N. polyvora* infection of *C. glomeratus* could significantly increase overwintering mortality. However, subtle effects of this type could only be discovered by carefully scrutinizing host-pathogen-parasitoid interactions.

CONCLUSIONS

In summary, the conclusions, based on the published data from tests designed to measure impact of microbial pest control agents on non-target predators and parasites, are listed below

- (1) Effects from indirect mortality of natural enemies consuming their

microbially infected hosts are probably more significant across all microbial types than the direct mortalities to predators and parasites.

- (2) Effects from sub-lethal factors or possibly mortality factors operating with longer time delays than usually evaluated in side effects tests (typically, 48 h) are more important than more immediate, direct mortalities for most natural enemies tested with microbials (particularly with protozoans).
- (3) Direct mortality to arthropod predators and parasites can be caused by bacterial and protozoan microbial pesticides.
- (4) No direct mortality from viruses of pests has been noted for arthropod natural enemies in the literature.
- (5) The direct effect of fungi on arthropod natural enemies has not been well studied and probably has been underestimated. Future studies should focus on mortalities that operate with longer time delays and when the insect is in a particularly favorable environment for fungal development (e.g. overwintering hibernacula).
- (6) There are probably many more stable interactions between natural enemies and entomopathogens in infected hosts than have been documented to date as compared to those known interactions between beneficials and chemical pesticides. This is because most entomopathogens are naturally occurring and natural enemies have been adapting mechanisms to reduce competition with them over evolutionary time (consider such adaptations as discrimination of infected hosts, fungistatic properties in teratocytes of braconids, etc.).
- (7) Standard methods need to be developed for microbial assays and for expressing more precisely the dosage administered to the host or natural enemy.
- (8) Duration of the tests should be relatively longer than standard bioassays.
- (9) The method of exposure is important and, with the exception of fungi, high mortality is generally encountered only when beneficial insects ingest the microbial.
- (10) In pest management, indirect mortality could be greatly reduced if the microbial was applied after parasitoids had infected the host.
- (11) Extensive research has gone into the study of basic interactions between entomophagous insects and pests of agricultural crops over the past 2 decades (under the banner of IPM). In this effort, much basic understanding of multiple trophic level species interactions in relation to MPs has been developed. We feel that these systems are ideal models for evaluating the impact of biotechnologically modified agents, especially microbial products on natural biological systems. In light of these possibilities, we recommend that greater research effort to standardize and develop assessment methods to measure impact of MPs on arthropod natural enemy systems be undertaken by researchers and sponsored by appropriate private and public agencies.

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